Balantidium Coli and its Action in the Intestine.—JANOWSKI (*Zeitschrift für klin. Med.*, 32, p. 415) reports the case of a man with severe chronic diarrhœa. There was no evidence of dysentery, malaria, or tuberculosis. The symptoms began three days after an excessive meal, including a great deal of pork and various kinds of sausage, followed by a large quantity of water. Opium and bismuth had no effect, unless to increase the diarrhœa. There were six to eight large stools daily, without pain, but with borborygmi, and followed by a dull pain in the anal region. In the examination of the feces, enormous numbers of balantidium coli were found. The patient was given quinine by the mouth and per enema for five days, by which time the parasites disappeared entirely, and, the other symptoms being much less severe, the patient left the hospital. That the parasites caused the diarrhœa in the beginning the author does not claim, though it can hardly be doubted that they had an important part in maintaining the chronic course.

Janowski gives an account of the salient points in the history of balantidium coli. Since the first report of Malmsten, forty years ago, only fifty-five cases have been published. Thirty-seven of these occurred in Scandinavia, Finland, and Russia. There was diarrhœa in all but one of the cases, but its relation to the parasites is made probable in only twenty-five, and in one case even large numbers were present without any apparent effect.

On the whole, however, the parasites when present in human intestines are to be looked on as unwelcome or even dangerous guests, and are likely to be associated with obstinate diarrhœa. In many cases the infection was noted in persons who had much to do with swine, and since the balantidium occurs almost constantly as a harmless parasite in these animals, the relation seems very close. Janowski also thinks contaminated drinking-water a not improbable source. In treatment, either quinine, as used by the author in the case now reported, or salicylic acid internally and in enemata containing one gramme to the litre, should be given for several days or until the stools are free from parasites.

Alpha-naphthol as a Reagent for Free Hydrochloric Acid.—F. WINKLER (*Centralblatt für inn. Med.*, 1897, No. 39) recommends a new test for free hydrochloric acid, which is really a reversal of Molisch's modification of a well-known furfurol reaction, and used by the latter as a test for glucose. Winkler uses either a 5 per cent. alcoholic or 10 per cent. chloroform solution of alpha-naphthol. To a small quantity of the suspected fluid a few granules of dextrose and a few drops of the naphthol solution are added. This is carefully heated. If hydrochloric acid is present a bluish-violet color appears, rapidly turning darker. Overheating masks the reaction. The grape-sugar may be added to the naphthol solution in the proportion of ½ to 1 per cent., or one can use milk-sugar or even milk. Hydrochloric acid in the proportion of 0.04 per thousand may be detected by this method. It does not respond to lactic or acetic, but does to sulphuric or phosphoric acid. In testing stomach-contents the reaction may occur without the addition of dextrose from dextrose found in the stomach from starch.

Acute Yellow Atrophy and Cirrhosis of the Liver.—VON KAHLDEN (*Münch. medicinische Wochenschrift*, October 5, 1897) believes that cases of acute yellow atrophy of the liver which are not rapidly fatal develop into typical examples of cirrhosis of the liver. Before dealing with this point, he reviews briefly the causes of acute yellow atrophy.

Whereas pregnancy is considered an important predisposing etiological factor, only a small proportion of the recently published cases show this association.

Von Kahlden points out the relationship between acute yellow atrophy and epidemics of icterus, and mentions Meder's fatal case of acute yellow atrophy occurring in a soldier, who was one of fifty men who had suffered from jaundice in a barracks epidemic. Many other similar observations have been recorded.

Various poisons, of which the more important are phosphorus and arsenic, may cause the disease.

Of special interest are those cases which follow various infectious diseases. It has also been observed after osteomyelitis, erysipelas, diphtheria, phlegmon, septic gangrene, and streptococcus injections. The onset of acute yellow atrophy during the later stages of syphilis has been comparatively frequently observed. Meder states that eighteen or twenty of the reported cases have followed syphilis.

The association between infections with the bacterium coli communis and icterus or acute yellow atrophy has been more than once pointed out, particularly by French observers. Kamén found the bacterium coli communis in various internal organs in five fatal cases of Winckel's disease occurring in an epidemic in which six infants were attacked. Stroebe recently found the colon bacillus in the liver of a girl, aged twenty-one years, who died of acute yellow atrophy on the seventh day of the disease. It was proved in this case that the infection was through the portal vein.

Von Kahlden reports a similar case in a woman, aged twenty-eight years, who became suddenly ill on May 31, 1895, the patient dying on June 9th of acute yellow atrophy of the liver. Cover-slip preparations from the liver showed very numerous short bacilli with rounded ends, which were proved by the cultures to be the bacillus coli communis.

The subsequent changes in the liver in cases where acute yellow atrophy does not rapidly terminate fatally are then dealt with. Marchand found that the liver of a woman, who had six months previously had an attack of acute yellow atrophy, showed both macroscopically and microscopically a picture very closely resembling cirrhosis of the liver. Stroebe reports a similar case showing marked evidences of cirrhosis only four weeks after the onset of the acute yellow atrophy. Von Kahlden relates a case of his own occurring in a woman, thirty-four years of age, whose illness began with an osteomyelitis of the sternum leading to spontaneous fracture. Following the osteomyelitis a general staphylococcus infection developed. During the course of the illness, and three and a half months previous to death, the patient had marked jaundice, which was attributed to the occurrence of acute yellow atrophy of the liver. The liver presented at the autopsy, both macroscopically and microscopically, a picture very similar to that seen in cirrhosis of the liver. Von Kahlden believed that in this case there had first been an acute yellow atrophy of the liver followed by cirrhosis, which he believes is the usual course in those cases where the disease is not rapidly fatal.